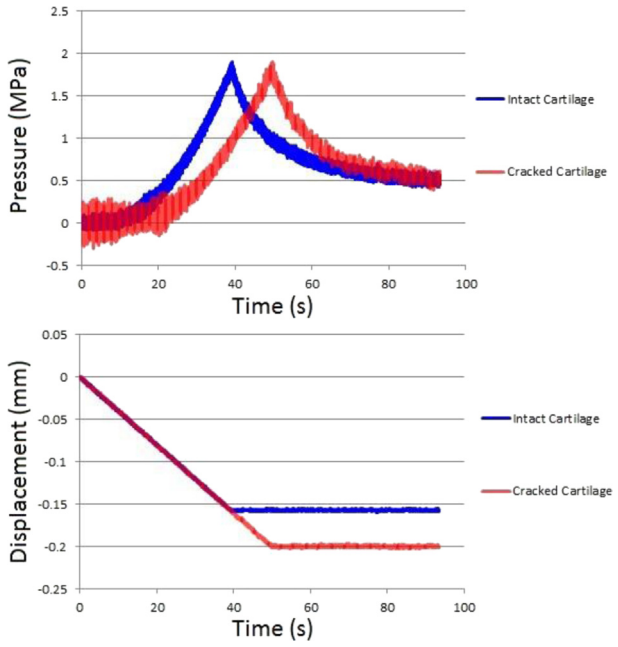

Figure 3. Comparison of stress relaxation curve in intact cartilage vs. cracked cartilage at compression speed of 4 μm/s until reaching a peak pressure of 2 MPa.

Histological analysis revealed that chondrocytes clusters in the radial zone show compression in the vertical direction and realignment of their long axis from the natural vertical orientation to an oblique pattern pointing away from the center of compression site. On Linear Polarized Microscopy (LPM), extensive collagen fiber reorientation is apparent (Figure 4), giving the impression that collagen fibers organize in two populations: 1- big pillars that can be seen in uncompressed and compressed intact (non-cracked) cartilage, and 2- small and free movable bundle groups that become more apparent under compression and reorient at 45° to the surface of the cartilage. Data collection is still in progress.

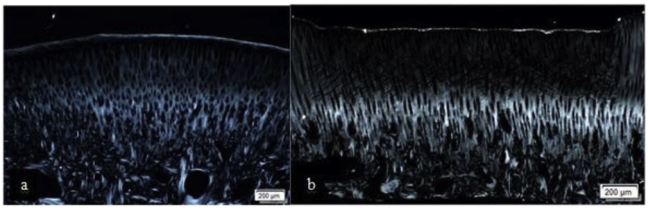

Figure 4. Rabbit patella viewed under LPM. a, Uncompressed patella with the bright part of extracellular matrix representing collagen fibers running parallel or perpendicular to the cartilage surface. b, Compressed patella showing the reoriented collagen fibers that appear as fine black lines running oblique to the cartilage surface, note the black lines run superimposed over the bright background that is similar to the one appearing in the unloaded cartilage.

We expect that stress-relaxation will be faster when the split lines are perpendicular to the crack and when cracks are made at smaller angles relative to the surface of the cartilage. Deformations of cracked edges and the extent of area affected by the loading will be identified too.
Conclusions: Even it is still early to draw conclusions from our preliminary results, it appears that the presence of a crack increases the amplitude of deformation applied to chondrocytes while lowering the time of high interstitial fluid pressure applied to chondrocytes by a given load, which may be significant to chondrocytes metabolism. The organization of collagen fibers may indicate that the big pillar fiber bundles limit the overall deformation of cartilage while the small bundles reduce the amount of deformation transmitted to chondrocytes. Structural changes in surface zone cartilage are among the earliest signs of very early OA. Understanding these changes in mechanics and cell signaling may allow altering the time course of OA by either mechanical intervention to normalize cell deformation and signaling in early OA, or pharmacological intervention to neutralize catabolic enzymes known to be released in chondrocytes near tissue cracks.

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ASSOCIATIONS BETWEEN POPLITEAL ARTERY WALL THICKNESS AND KNEE STRUCTURE IN ADULTS WITHOUT CLINICAL KNEE DISEASE: A PROSPECTIVE COHORT STUDY

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Purpose: There is evidence for a vascular contribution to the pathogenesis of osteoarthritis. Our aim was to use an asymptomatic cohort to examine the association between popliteal artery wall thickness, previously shown to be associated with risk of generalized osteoarthritis, and knee structural changes.
Methods: 297 adults with no significant knee pain, injury, or history of clinical knee disease were recruited. Participants underwent knee magnetic resonance imaging at baseline and 2 years later. Popliteal artery wall thickness, knee cartilage volume and bone marrow lesions (BML) were assessed.
Results: Of 278 participants with valid popliteal artery wall thickness measurement, 254 (91.4%) completed 2-year follow-up. After adjusting for age, gender, body mass index and tibial bone area, increased popliteal artery wall thickness was associated with reduced medial tibial cartilage volume ($B = -6.7$, 95% CI −12.9, −0.6, $p = 0.03$) and increased rate of medial tibial cartilage volume loss ($B = 0.06$, 95% CI 0.01, 0.12, $p = 0.03$). There was a trend for medial tibiofemoral BML deterioration in relation to increased popliteal artery wall thickness (odds ratio=1.07, 95% CI 0.99, 1.15, $p = 0.07$). No significant associations were observed with lateral tibiofemoral compartment.
Conclusion: Increased popliteal artery wall thickness was associated with adverse changes in knee structure, as evidenced by reduced medial tibial cartilage volume, increased rate of cartilage volume loss and a trend for BML deterioration over 2 years. These findings suggest an association between vascular pathology and early knee structural changes, supporting the hypothesis that vascular health may play a role in the development of knee osteoarthritis.

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ASSOCIATIONS BETWEEN LEVELS OF URINARY C-TELOPEPTIDE FRAGMENTS OF TYPE II COLLAGEN AND KNEE STRUCTURE IN MIDDLE-AGED WOMEN WITHOUT CLINICAL KNEE DISEASE

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Purpose: There is evidence for an association between levels of urinary C-telopeptide fragments of type II collagen (uCTX-II) and risk of knee osteoarthritis. The aim of this study was to examine the association between uCTX-II levels and knee cartilage and bone changes in middle-aged women without clinical knee disease.
Methods: 140 women, aged 40 – 67 years, with no significant knee pain, knee injury or any forms of arthritis, underwent knee MRI at baseline and two years later. Cartilage volume, cartilage defects, tibial plateau bone area and bone marrow lesions (BMLs) were measured using validated methods. Baseline uCTX-II was measured using ELISA.
Results: A higher baseline uCTX-II level was associated with increased prevalence of medial tibiofemoral cartilage defects (OR 4.36, 95%CI 1.58–12.04), greater medial (regression coefficient 80.2, 95%CI 9.3–151.1) and lateral (regression coefficient 86.0, 95%CI 33.3–138.7) tibial plateau bone area, and increased prevalence of lateral tibiofemoral BMLs (OR 10.62, 95%CI 1.82–61.85). Baseline uCTX-II levels were not significantly associated with baseline tibial cartilage volume or changes in knee cartilage (volume and defects) or bone (bone area and BMLs) over two years.
Conclusion: In middle-aged women without clinical knee disease, higher uCTX-II levels were associated with detrimental changes in knee cartilage and bone cross-sectionally but not over two years. This suggests that uCTX-II may be a sensitive biomarker of early structural features of knee osteoarthritis. Further work will be needed to determine its sensitivity to change and whether it predicts progression of disease over longer time periods.